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THE TAKE-ALL DISEASE OF CEREALS AND GRASSES CAUSED BY OPHIOBOLUS CARICETI (BERKELEY AND BROOME) SACCARDO

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THE TAKE-ALL DISEASE OF CEREALS AND GRASSES CAUSED BY OPHIOBOLUS CARICETI (BERKELEY AND BROOME) SACCARDO[1]

ROBERT S. KIRBY

This disease, long known in Europe, Japan, and Australia, was first correctly reported from America in 1920 (Kirby and Thomas, 1920). Reports of the finding of take-all in Illinois appeared as early as 1919 (Humphrey and Johnson, 1919, and Humphrey, Johnson, and McKinney, 1921) but this was a case of mistaken identity, further investigations (McKinney, 1923) having shown that disease to be distinct from take-all. It is now known as the rosette disease of wheat.

Because of the severity of take-all in certain foreign countries, much concern has been expressed lest it appear in the United States and cause heavy losses to cereals. Since the value of wheat, oats, rye, and barley in the State of New York in 1919, according to the fourteenth census, was equal to about that of one-ninth of all crops grown, or nearly equal to the value of the fruit or the potato crop in the State, the discovery of the disease here seemed to warrant a careful study of its distribution and severity under New York State conditions. The present investigations cover a period of four years during which time the disease has been studied in the field and greenhouse.

HOST RANGE

As far as is known, take-all is restricted to the Gramineae. An examination of the data given in table 1 shows that of the two subfamilies of the Gramineae, fewer members of the Panicoideae appear to be subject to the disease than of the Poacoideae. Of these species which were investigated, two out of fourteen in the Panicoideae, and eighty-three out of one hundred and forty-two in the Poacoideae, are susceptible. In the Panicoideae, susceptible species are found in only one of the three tribes investigated; in the Poacoideae all of the seven tribes tested contain forms liable to infection.

The tribe Hordeae seems to contain more susceptible species than any other tribe in the Gramineae. The other tribes, arranged according to the number of susceptible species in them, are as follows: Agrostidae, Festuceae, Phalarideae, Avenae, Oryzeae, Paniceae, and Chlorideae.

In no tribe were all of the species found to be susceptible, but all of the species inoculated in a few of the genera — Agropyron, Elymus,

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Hordeum, Hystrix, Phleum, and Stipa — became affected. Of all the genera tested, the fewest resistant species were found in Agropyron, Elymus, Hordeum, Hystrix, Stipa, and Triticum. Of these, the species in Triticum seem to be the most susceptible since they are the only hosts to show any marked degree of hypoplasia.

RELATIVE SUSCEPTIBILITY OF GRASSES AND OF CEREALS

Grasses

The most important rôle of the grass hosts, excluding the cereals, is as harborers of the pathogene. Since the importance of the individual host in this respect depends upon the number of perithecia produced on it, the evidence presented in table 1 is given in terms of the relative number of perithecia. The number of perithecia was determined for each species by dissecting numerous culms under the binocular. On all grasses reported as hosts, typical perithecia and ascospores have been found.

The host-range tests made in the greenhouse and tabulated in table 1 represent a repetition and extension of those previously reported by the

TABLE 1. HOST RANGE OF *Ophiobolus cariceti* AS OBSERVED FROM INOCULATION TESTS IN THE GREENHOUSE, OBSERVATIONS IN THE FIELD, AND REPORTS OF OTHER INVESTIGATORS

Name of grass *	Relative number of perithecia on plant †		Presence of perithecia previously recorded ‡
	In greenhouse tests	In the field	
Agropyron sp.	.....	.....	(1)
caninum (L.) Beauv.	Many.....	.....	(14)
cristatum J. Gaertn.	Many.....	.....	(14)
desertorum (Fisch.) Richt.	Very many	.....	.....

* All the grasses in this test were grown from seed obtained from the following sources. Dr. E. B. Mains, Purdue University, LaFayette, Indiana; Professor A. H. Larson, Seed Laboratory, University of Minnesota, St. Paul, Minnesota; Berkeley, California, grass gardens of the Cereal Office of the United States Department of Agriculture.

† Few=less than ten perithecia per culm.
Moderate=from ten to forty perithecia per culm.
Many=more than forty perithecia per culm.

‡ References to literature in last column:
1=Saccardo (1875)
2=Waters (1920a)
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TABLE 1 (*continued*)

Name of grass *	Relative number of perithecia on plant †		Presence of perithecia previously recorded ‡
	In greenhouse tests	In the field	
Agropyron (*continued*) :			
intermedium (Host.) Beauv	Many	.....	(14)
obtusiusculum Lange	Moderate	.....	.....
repens (L.) Beauv	Many	Moderate	(2)
richardsoni Schrad	Many	.....	.....
scabrum (R. Bv.) Beauv	.....	.....	(3)
smithii Rydb	Moderate	.....	(14)
spicatum (Pursh) Rydb	Many	.....	.....
tenerum Vasey	Many	.....	(14)
Agrostis canina L	None	.....	.....
palustris Huds	None	Few	.....
(=*A. alba* L.)	.....	.....	.....
perennans (Walt.) Tuckerm	Few	.....	.....
retrofacta Willd	Few	.....	.....
stolonifera L	None	.....	.....
verticillata Vill	Few	.....	.....
Aira caespitosa L	.....	.....	(4)
caryophyllea L	None	.....	.....
Alopecurus geniculatus L	Moderate	.....	.....
pratensis L	None	.....	.....
Andropogon scoparius Michx	None	.....	.....
Anthoxanthum odoratum L	Many	.....	.....
Arrhenatherum elatius (L.) Beauv	None	.....	.....
Avena brevis Roth	None	.....	.....
fatua L	None	.....	.....
nuda L. (hull-less oats)	None	.....	.....
orientalis Schreb. (side oats)	None	.....	.....
sativa L. (common oats)	None	None	(5)

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TABLE 1 (*continued*)

Name of grass *	Relative number of perithecia on plant †		Presence of perithecia previously recorded ‡
	In greenhouse tests	In the field	
Avena (*continued*) :			
sterilis L.	None	.....	.....
strigosa Schreb	None	.....	.....
Beckmannia erucaeformis (L.) Host	Many	.....	.....
Bouteloua curtipendula (Michx.) Torr	None	.....	.....
gracilis (HBK.) Lag	None	.....	.....
Briza maxima L.	None	.....	.....
Bromus arvensis L.	Moderate	.....	(14)
brizaeformis Fisch. & Mey	Moderate	.....	.....
carinatus Hook. & Arn	Few	.....	.....
ciliatus L.	Many	.....	(14)
erectus Huds	Moderate	.....	(14)
exaltatus Bernh	Many	.....	.....
hordeaceus L. (=*B. mollis* L.)	Moderate	.....	(3)
inermis Leyss	None	.....	.....
japonicus Thunb	Few	.....	(14)
lanuginosus Poir	Many	.....	.....
madritensis L.	Moderate	.....	(14)
marginatus Nees	Moderate	.....	.....
pendulinus Sesse	Few	.....	.....
pumpellianus Scribn	Moderate	.....	.....
racemosus L.	Moderate	.....	(14)
rigidus Roth	Moderate	.....	.....
secalinus L.	Moderate	.....	(14) (7)
sterilis L.	Many	.....	(6)
subvelutinus Shear	Moderate	.....	.....
tectorum L.	Many	.....	(14)
tectorum nudus Klett & Richter	Many	.....	.....

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TABLE 1 (*continued*)

Name of grass *	Relative number of perithecia on plant †		Presence of perithecia previously recorded ‡
	In greenhouse tests	In the field	
Bromus (*continued*):			
unioloides (Willd.) HBK	None	.....	.....
villosus Forsk	None	.....	.....
Calamagrostis canadensis (Michx.) Beauv	None	.....	.....
Capriola dactylon (L.) Kuntze	None	.....	.....
Cenchrus echinatus L	None	.....	.....
tribuloides L	None	.....	.....
Chaetochloa geniculata (Lam.) Millsp. & Chase	.....	.....	(7)
lutescens (Weigel) Stuntz	None	.....	.....
Chloris virgata Swartz	None	.....	.....
gayana Kunth	None	.....	.....
Cynodon sp	.....	.....	(1)
Cynosurus cristatus L	None	.....	.....
Dactylis glomerata L	None	.....	.....
Danthonia intermedia Vasey	None	.....	.....
Deschampsia elongata (Hook.) Munro	None	.....	.....
Digitaria sanguinalis (L.) Scop	None	.....	.....
Echinochloa crus-galli (L.) Beauv	None	.....	.....
frumentacea (Roxb.) Link	None	.....	.....
Elymus arenarius L	Many	.....	.....
australis Scribn. & Ball	Many	.....	(14)
canadensis L	Many	.....	(14)
condensatus Presl	Many	.....	.....
curvatus Piper	Many	.....	.....
europaeus L	Many	.....	.....
giganteus Vahl	Few	.....	.....
hirsutiglumis Scribn. & Sm	Few	.....	.....
sibiricus L	Moderate	.....	.....

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TABLE 1 (*continued*)

Name of grass *	Relative number of perithecia on plant †		Presence of perithecia previously recorded ‡
	In greenhouse tests	In the field	
Elymus (*continued*):			
virginicus L.	Many		(14)
Eragrostis lugens Nees	None		
Festuca bromoides L.	None		
elatior L.	Few		(14) (7)
heterophylla (Lam.) Hack.	None		
megalura Nutt.	None		
octoflora Walt.			(7)
ovina L.	None		
ovina tenuifolia (Sibth.) Sm.	None		
pratensis Huds.	None		
rubra L.	None		
Gastridium lendigerum (L.) Gaud.	Few		
Glyceria grandis Wats.	None		
nervata (Willd.) Trin.	None		
Holcus lanatus L.	Few		
Hordeum distichon L.	Moderate		
jubatum L.	Many		(14)
murinum L.	Few		(8)
nodosum L.	Many		
pusillum Nutt.	None		(7)
secalinum Schreb.	Many		
spontaneum C. Koch	Many		
vulgare L. (barley)	Many	None	(9)
Hystrix patula Moench	Many		(14)
Koeleria cristata (L.) Pers.	Many		
Lolium multiflorum Lam.	None		
perenne L.	None		

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TABLE 1 (*continued*)

Name of grass *	Relative number of perithecia on plant †		Presence of perithecia previously recorded ‡
	In greenhouse tests	In the field	
Lolium (*continued*):			
subulatum Vis.	None	.....	.....
temulentum L.	Few	.....	(14)
Oryza sativa L.	None	.....	(9)
Oryzopsis miliacea Benth. & Hook.	Many	.....	.....
Panicum virgatum L.	Few	.....	.....
Paspalum setaceum Michx.	None	.....	.....
Pennisetum villosum R. Br.	None	.....	.....
Phalaris arundinacea L.	Few	.....	(14)
bulbosa L.	Few	.....	.....
caroliniana Walt.	None	.....	.....
minor Retz.	None	.....	.....
Phleum pratense L.	Few	Few	.....
Phragmites communis Trin.	None	.....	.....
Poa alpina L.	Few	.....	.....
annua L.	None	.....	.....
caesia Sm.	None	.....	.....
chaixii Vill.	Few	.....	.....
compressa L.	Moderate	Moderate	.....
palustris L.	Moderate	.....	.....
pratensis L.	Few	.....	.....
Polypogon littoralis Sm. (With)	None	.....	.....
maritimus Willd.	None	.....	.....
monspeliensis (L.) Desf.	Moderate	.....	.....
Poppophorum vaginatum Buckl.	None	.....	.....
Puccinellia simplex Scribn.	None	.....	.....
Secale cereale L. (rye)	Few	Few	(10)
Sesleria coerulea (L.) Scop.	None	.....	.....

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TABLE 1 (*concluded*)

Name of grass *	Relative number of perithecia on plant †		Presence of perithecia previously recorded ‡
	In greenhouse tests	In the field	
Sorghum sudanense (Piper) Stapf	None	.....	.....
Sporobolus interruptus Vasey	None	.....	.....
Stipa capillata L.	Moderate	.....	.....
lepida Hitchc.	Moderate	.....	.....
pennata L.	Few	.....	.....
spartea Trin.	Moderate	.....	.....
viridula Trin.	Few	.....	.....
Triodid flava (L.) Hitchc.	None	.....	.....
Triticum aegilopoides Bal. (wild small spelt)	Many	.....	.....
compactum Host (club wheats)	Many	None	(11)
dicoccoides Körn. (wild emmer)	Heavy	.....	(14)
dicoccum Schr. (emmer)	Many	Many	(14)
durum Desf. (durum wheats)	Many	None	(13)
monococcum L. Einkorn	Few	None	(14)
polonicum L. (Polish wheat)	Moderate	.....	(14)
spelta L. (large spelt)	Moderate	Moderate	(14)
turgidum L. (poulard wheats)	Many	None	(14)
vulgare Vill. (common wheats)	Many	Many	(12)
Zea mays L. (field corn)	None	.....	.....

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writer (Kirby, 1922). The method used was essentially the same, except that sterilized soil was used throughout and the inoculum consisted of many more pure-culture isolations. These tests were started on January 3 and 4, 1922, and continued for a period of one year.

Six species of grasses — *Anthoxanthum odoratum*, *Bromus hordeaceus*, *Bromus villosus*, *Holcus lanatus*, *Poa compressa*, and *Poa pratensis* — which

were reported as immune in the first tests, were found during the second tests to be susceptible to the take-all pathogene. All grasses found to be hosts in the first series of tests became affected again in the second.

The grasses reported as hosts under field conditions were, in every case, found in wheat fields infested with take-all. Infected *Agropyron repens* was observed in nearly all of the 137 take-all-infested wheat fields visited in 1922 and 1923 and this seems to be the most susceptible wild grass in New York. *Poa compressa* was found infected in Tompkins County only, where it was almost as heavily infected as *Agropyron repens*. *Bromus secalinus*, *Agrostis palustris*, and *Phleum pratense* were found to be infected in Cayuga, Wayne, and Tompkins Counties, but never more than one or two perithecia could be found on a plant. Under field conditions in New York State, however, the grasses appear to be tolerant to the take-all organism, since they are rarely severely stunted even when heavily infected.

Cereals

Apparently take-all is restricted to the small grains. Concerning their relative susceptibility, Hori (1901), reporting on the situation in Japan, states that barley and rye are slightly more susceptible than wheat since they exhibit a greater degree of stunting and have their yields reduced more than does wheat. Oats are reported in Australia (Anonymous, 1919) as being less affected than wheat and barley, but not immune, while Richardson (1911) reports that oats are not affected by take-all. Rice has been reported by Hori (1901) and Tanaka (1917) as being very slightly susceptible to take-all.

The present investigations indicate that spring-planted barley and oats growing under field conditions either escape or are immune to take-all, and that spring-planted wheat becomes infected only under conditions which are extremely favorable to the pathogene. Rye is apparently very much more resistant than wheat. When winter rye and ten varieties of winter wheat were grown in a field test in 1922 to determine their relative susceptibility, 87 per cent of the rye plants and 96.6 per cent of the wheat plants were diseased. Among these diseased plants there was no stunting of the rye plants, but 75.6 per cent of the wheat plants were stunted. This difference in resistance is further borne out by the fact that in badly infested wheat fields volunteer rye is very seldom diseased.

Under greenhouse conditions wheat was the most susceptible of the cereals; barley was more resistant that wheat but less resistant than rye, which was very slightly affected; oats and rice appeared to be immune.

It seems that under New York conditions winter wheat is the only cereal which will be seriously damaged by take-all. However, in warmer sections of the United States where winter barley is grown, the disease may cause a considerable loss to that crop. Rye will probably never suffer any appreciable injury from the disease in New York. Spring-

planted cereals, apparently because of their shorter growing season, usually escape the disease, and therefore it is unlikely that they will ever suffer an appreciable loss from it.

VARIETAL SUSCEPTIBILITY IN WHEATS

No variety of wheat has been found which is immune to take-all, although there seem to be marked varietal differences in susceptibility. Pridham (1919) found on testing eighty-one varieties that the percentage of badly infected plants varied from 1 to 33.7 per cent. Previous workers agree that red wheats are more resistant than white wheats. In the Union of South Africa (Anonymous, 1922 a), take-all is said to attack the hard durum types more than the soft *vulgare* wheats. Pridham (1919) reports early-maturing wheats to be the most nearly free from the disease in Australia, while in England (Anonymous, 1913) and in New Zealand (Waters, 1920 a) early or short-season varieties are held to be the most susceptible. Spring wheats are reported as escaping the disease (Foex, 1919 b).

In a previous paper (Kirby, 1922) the writer reported tests with sixty-two varieties of wheat for relative susceptibility under greenhouse conditions. These tests showed that common, or *vulgare*, wheats exhibit about the same degree of susceptibility whether they are of spring or winter habit, soft or hard, and red or white. Of the various kinds of wheat the durum and poulard wheats were all heavily infected, and these are probably the most susceptible varieties. The club, emmer, and common wheats varied in their degree of infection from heavy to moderate, and were more resistant than the durum and poulard wheats. The Polish and spelt wheats were only moderately infected, and these are apparently more resistant than the common type. Einkorn seems to be the most resistant, since it was only slightly infected.

In order to determine the relative susceptibility of a few winter wheats grown in New York, ten varieties were planted in triplicate plots in the fall of 1921. In the bottom of each drill row in one of the three plots, an equal amount of pure culture of the take-all organism growing on sterilized wheat kernels was placed at the time of planting. At the same time an equal amount of take-all-infested soil and infested parts of wheat plants, respectively, were added to each row in the other two plots.

In July, 1922, when the wheat had matured, the plants of each variety were dug and data were taken on the percentage of infected and of healthy plants, and the degree of stunting. Then, for the healthy plants and for each of the three degrees of stunting, data were taken on the average number of heads per plant and the average weight and number of seeds produced per head. The average yield per plant was obtained by multiplying the average number of culms by the average weight of seeds produced per head. By this method it was hoped that reduction in yield

by take-all would be determined for each variety. However, in most varieties few disease-free plants were found. Therefore it was impossible to determine accurately the reduction in yield for individual varieties. But the data when averaged for the ten varieties gave fairly accurate figures, and showed that the yield of a take-all-infected plant which is not stunted is reduced 50.2 per cent as compared with that of a healthy plant; that the yield of a plant which is stunted less than half of its normal height is reduced 90.77 per cent; and that the yield of a plant dwarfed to more than half its normal height is reduced 99.39 per cent. The percentage of plants of each variety in each of the above classes, and the average yield in grams of one hundred plants of each variety, are given in table 2. The data in this table show that there is a marked difference in the degree of susceptibility of various wheat varieties. This difference

TABLE 2. RELATIVE INJURY AND YIELDS OF TEN WHEAT VARIETIES

	Per cent of plants infected with take-all			Per cent of plants not infected	Average yield of 100 plants (grams)
	Plants less than $\frac{1}{2}$ normal height	Plants $\frac{1}{2}$ to $\frac{9}{10}$ normal height	Plants of normal height		
Forward	11.0	21.1	66.7	1.2	135.4
Dawson	21.2	14.4	57.3	7.1	118.0
Red Wave	11.2	21.5	61.9	5.4	103.6
Golden Cross x New Columbia	23.7	22.8	49.7	3.8	112.9
Gold Coin (No. 6 Jr.)	22.3	28.1	45.4	4.2	92.3
Currells Prolific x Fultz	38.7	18.1	43.2	.....	70.4
Fulcaster	23.3	22.0	53.2	1.5	74.78
Red Rock	39.1	18.9	40.8	1.2	68.50
Cosgrove	32.8	41.4	23.9	1.9	40.40
P 1068 Kanred	47.4	23.0	25.4	4.2	30.00
Average	27.07	23.13	46.75	3.05	84.63

seems to be constant, since practically every variety held the same relative position of susceptibility in each of the plots. The experiment was repeated in 1923, with duplicated plots. The results substantiated those of 1922 concerning the relative susceptibility of the ten varieties previously used. The testing of additional varieties showed that P 1066, Kanred Ks. 2401, and Turkey, were similar to P 1068 in that they all were very susceptible. Black Winter Emmer showed about the same degree of resistance as Dawson, and Red Winter Speltz and White Beardless Speltz showed greater resistance than any of the other wheats.

Data based only on the percentage of plants escaping take-all infection are apparently worthless, since under favorable conditions nearly all plants become infected before reaching maturity. The varieties that yield the highest are invariably those in which the largest percentage of the infected plants are able to develop to normal size. In the varieties previously mentioned it will be noted that the harder wheats are the more susceptible and that there is seemingly no difference in susceptibility between red and white wheats.

THE DISEASE

NAMES

The name *take-all* has been generally applied in Australia, England, and other English-speaking countries to the disease under consideration. In the early stages it is called *yellow-leaf disease* and *take-all*. In later stages it is called *white heads*, *wit-roest*, *stand dead disease*, *white wilt*, *habit*, *wilt*, and *smoky*. In Europe somewhat similar diseases are frequently referred to as *foot rot*, *black foot*, *black stem*, *stalk disease*, *foot disease*, *root disease*, *black leg*, *straw blight*, *pietin*, and *pietin du ble*, but these are not to be confused with take-all.

HISTORY AND RANGE

Early references to root diseases of cereals are of little value, since it is impossible to determine whether take-all or a similar disease is meant. The take-all organism was first described by Berkeley and Broome (1861) from specimens found at Batheaston, England, on *Aira caespitosa*. Saccardo (1875) recorded the fungus from Italy on *Agropyron* sp. and *Cynodon* sp. In 1878 he reported it from France on wheat. It was again found in England in 1884 (Anonymous, 1913), and was next reported from France by Prillieux and Delacroix (1890). In 1901 it was recorded from Japan by Hori (1901), who stated that the disease had been present there since 1890 or 1891. McAlpine (1904) reported the disease as widespread in Australia at that time, and stated that it had been known there since 1852. Lindau (1908) records the presence of the disease in Germany and Holland. Dombrovski (1909) states that take-all occurs in Russia and Denmark, and Mangin (1914) records Belgium and Portugal as having take-all-infected cereals. The disease is reported by Waters (1920, a and b) as having been present in New Zealand for many years. It has been reported also from the Union of South Africa (Anonymous, 1922 a), where it was first found in 1904. In December, 1923, Dr. W. P. Fraser informed the writer that take-all had been found during the previous summer in a field of Marquis wheat in Saskatchewan, Canada.

Take-all was first reported from the United States in 1920 (Kirby and Thomas, 1920). In August of the next year, Humphrey, Johnson, and

McKinney (1921) called attention to reports of its appearance in Arkansas and Oregon. The writers state also that a "similar" trouble, of which investigations have indicated "a difference cause," has appeared in Virginia. Later, in a numeration of the various collections of take-all which have been made, McKinney (1923) alludes to the fact that the material collected in Virginia was in reality take-all, perithecia having apparently been discovered subsequently by members of the staff of the

FIGURE 1. LOCATION OF ALL INFESTED CEREAL FIELDS FOUND DURING THE 1921, 1922, AND 1923 SURVEYS

Office of Cereal Investigations, United States Department of Agriculture. In 1922 take-all was reported from Indiana and from additional localities in New York (Kirby, 1922), and it was shown that the disease was present in Kansas.[2] In 1923 specimens of the fungus which were collected in Tennessee in May were identified by the writer.[3] In addition the disease was reported from California (Mackie, 1923), and during July of 1923 Dr. J. A. Clark informed the writer that a considerable area of wheat infested with take-all had been found in North Carolina.

[2] Reported to the writer in a letter from R. P. White dated 1922, also by cultures which were sent to the writer in May, 1922.

[3] These specimens were sent to the writer by Dr. C. D. Sherbakoff on May 21, 1923.

Surveys were made in 1921, 1922, and 1923 in cooperation with the Plant Disease Survey of the United States Department of Agriculture, to determine the distribution of take-all in New York During those three years the surveys extended over fifty-seven of the sixty-two counties in New York, and included all sections where spring or winter wheat is commonly grown. The location of all take-all-infested winter-wheat fields found during these three surveys is shown in the accompanying map (figure 1).

Plants affected with take-all were found in 205 out of the 538 winter-wheat fields surveyed. The 205 infested fields were located in eighteen counties in the west central part of the State. This infested area seems to have definite boundaries, and coincides closely with the principal winter-wheat-producing area of New York. Within these eighteen counties, 48 per cent of the fields surveyed during the years 1921 to 1923 were found to be infected.

ECONOMIC IMPORTANCE

Take-all is one of the most serious diseases of winter wheat in the countries where it occurs. Its importance varies from year to year, depending at first on the climatic conditions; but later, when an area is once infe ted, the importance of the disease seems then to depend largely on whether or not rotation is practiced. Continuous cropping always results in increasingly greater losses; for example, in the Union of South Africa, where this is the prevailing practice (Anonymous, 1922 a), take-all results in an enormous amount of damage, and losses up to 100 per cent are reported. Hardly a field is free from the disease and it spreads rapidly. It is said that wheat-growing in the infested area there will soon be a thing of the past unless rotation is adopted. In Australia, where crop rotation is the exception and not the rule, many fields are almost entirely useless for wheat; and Mackinnon (1920) states that take-all reduces the average yield of Australian wheat fields from 12 to 15 per cent in certain years, although the average reduction for all years is probably not more than 7 per cent annually. In the United States, Stakman (1922) says that at Hillsboro, Oregon, the loss is estimated at one-third of the crop, and it is further reported (Anonymous, 1922 b) that in the same year, in that State, a considerable number of fields were so damaged as not to be worth threshing. In New York where rotation is usually practiced, the loss in the infested area varies from 0.3 to 2 per cent, although losses as high as 20 per cent have been observed in individual fields where rotation was neglected.

This disease causes a marked dwarfing of all parts of the wheat. The effect of the different degrees of dwarfing on the varieties of winter wheat grown in duplicate plots at Ithaca are shown in table 3. With increased severity of the disease as indicated by the degree of dwarfing, there is a proportionate decrease in the number of heads per plant, the number of

TABLE 3. EFFECT ON YIELD OF TEN VARIETIES OF WINTER WHEAT GROWN IN TRIPLICATE PLOTS RESULTING FROM THE DIFFERENT DEGREES OF DWARFING CAUSED BY TAKE-ALL

Class and description	Average number of heads per plant	Average number of seeds per head	Weight of 100 seeds (grams)	Average yield per plant (grams)	Average per cent of reduction in yield caused by take-all
1 Plants with no take-all.....	3.43	25	3.45	2.875	0
2 Plants affected with take-all. No reduction in height.....	2.65	23.6	2.295	1.431	50.2
3 Plants affected with take-all. Somewhat stunted, but never less than one-half the height of healthy plants.....	1.75	13.7	1.11	0.266	90.77
4 Plants affected with take-all. Stunted so that they are less than one-half the height of healthy plants.....	0.43	6.4	0.637	0.0176	99.39

seeds per head, the weight of the seeds, and the yield per plant. The reduction in weight of the seeds means that the seeds from wheat plants which were stunted by the disease will be so shriveled that they will be worthless as grain and most of them will be so light as to be blown out with the chaff in threshing. In addition to the reduction in the grain yield, there is a very marked degree of reduction in the amount of straw produced. This reduction in the yield of straw was apparently as high as 20 to 25 per cent, on an average, for all affected wheat plants.

The amount of reduction in the average number of heads, the number of seeds produced, the weight of the seeds, and the yield per plant, did not vary appreciably between the plants of different varieties which were in the same class as to dwarfing or having the same degree of infection, and the difference in the amount of loss between two fields or varieties was due to different percentages of plants occurring in the four classes. Class 2, in which the affected plants were not stunted, contained 46.75 per cent of all the plants. Since there was 50.2 per cent reduction in yield in this class, the total theoretical yield which would have been realized if all the plants had been free of take-all was reduced 23.4 per cent. In the same way, for the 23.13 per cent of all plants in class 3,

where the affected plants were stunted not to exceed one-half the height of take-all-free plants, the reduction in yield was 20.9 per cent; and for the 27.06 per cent of all plants in class 4, where the affected plants were less than one-half as high as normal plants, the reduction was 26.90 per cent. Thus this disease caused an average reduction in yield in the ten varieties of winter wheat tested, of approximately 71.2 per cent. Since plants were not observed to die before the culms started to shoot up, the higher yield of plants recorded as disease-free seemingly was not due to the presence of fewer plants in a given area. In fact, there was evidence to show that the disease-free plants growing in the inoculated rows yielded less per plant than did the plants growing in check rows, where all the plants were free of disease. Thus it seems that the recorded loss due to this disease is probably too low rather than too high.

The 1922 survey in New York showed that 60 per cent of the infected plants in all fields examined were in class 2, 35 per cent in class 3, and 5 per cent in class 4. In applying the same rates of reduction in yield, the average loss in the infected plants would be 66.8 per cent. Since 1.1 per cent of the plants in infested fields were diseased, the loss in these fields was approximately 0.71 per cent.

SYMPTOMATOLOGY

The take-all disease affects the roots, the culms, the leaves, and the heads of its hosts, producing definite and characteristic symptoms. Signs[4] of the disease appear on the roots and on the lowest two or three internodes of the culms.

Symptoms

On roots.— Brown necrotic lesions appearing on the roots are the first symptoms of this disease. Such lesions were noticeable on the roots of winter wheat collected from a field on March 21, 1923, and the causal fungus was isolated from several of them. These lesions may completely or only partly surround the root. With the advent of warm spring weather the lesions increase in number, so that by the time the plant enters the jointing stage and sends up tillers (usually May 10 to 20 at Ithaca, New York) the greater number of the roots of badly infected plants are brown and dead and nearly all of the living roots have one or more lesions.

The killing of the roots progresses from the older to the newer ones, and to offset this loss the older roots continue to send out an abnormal number of branches which give them a woolly appearance (Plate II, A). New roots are often sent out from a point on the crown immediately above that of the emergence of the old ones. The killing of the roots weakens them to such an extent that a slight pull will break the plant off at or near the crown (Plate 1, B). Because of the fasciculation of the

[4] Glossary. *In* Laboratory outlines in plant pathology, by H. H. Whetzel, L. R. Hesler, C. T. Gregory, and W. H. Rankin. 1916.

roots a considerable amount of soil is often pulled up with them, giving them a characteristic clubbed appearance.

Under greenhouse conditions the take-all pathogene has been observed to cause lesions on the roots of wheat seedlings within fourteen days after planting, and to cause the death of the seedlings within another week.

On culms and heads.— The first symptoms to be observed in wheat plants under field conditions appear immediately after the plants have entered the jointing stage and when they are from six to ten inches high. These symptoms consist of a yellowing of the leaves and a dwarfing of the plants. (The earliest date of observing these symptoms at Ithaca is May 22.) The plants that are most severely affected soon die. By flowering time the diseased areas in the fields are strikingly characterized by the whitish color and the stunted stature of the plants contained in them. These areas are often 15 feet or more in diameter and are usually circular in outline. They contain from one to many plants. Their edges often overlap in badly infected fields, so that one may walk across such a field without leaving them. In some fields the disease occurs for several feet along the drill rows. The individual plants are usually dead and ashy white in color, and in the center of large areas are either killed out or dwarfed to a few inches in height. Most of the diseased plants are dead by flowering time, and on an average not more than one head per plant is developed.

The whitish color of the plant suggests the name *white heads* which is used by some writers to designate the later stage of this disease. A characteristic symptom is the marked reduction of the normal number of tillers, many of which die soon after their formation.

There is no striking change in the symptoms from the time of flowering to harvest. However, the diseased leaves and culms may become sooty due to the growth of saprophytic species of Cladosporium and Mucor. The " white head " stage only was observed in rye. Rye shows no chlorotic symptoms until after flowering, and therefore exhibits little or no stunting and only a moderate shriveling of the kernels.

The symptoms in barley as reported by Hori (1901) in Japan differ from those of wheat in that they often appear within thirty to forty days after the winter barley is sown. This stage is spoken of as the *yellow-leaf*. The other symptoms on barley are essentially like those on wheat.

On *Agropyron repens* the chlorosis and dwarfing were not conspicuous, but many lesions were found on the perennial rootstocks.

Signs

The first signs appear very soon after the chlorotic symptoms (May) and consist of a mat, or plate, of brown mycelium which extends from the root upward for several inches above the crown. The mycelium is in the leaf sheaths and between the culm and the inner leaf sheath, where it

occurs as a plate-like mass. It causes a discoloration of the first and second internodes, forming a dark brown or black zone at the base of the diseased culms. Perithecia are formed during June and July in comparatively large numbers on the stems and in small numbers on the roots. After the death of diseased plants, or rarely before, the beaks of the perithecia may protrude through the outer leaf sheath.

In rye the plate mycelium is less pronounced and fewer perithecia are developed than in wheat, while on the susceptible wild grasses a definite plate-mycelium is practically always lacking.

Comparative symptomatology

True take-all differs in its signs and symptoms from the so-called "take-all" of Humphrey, Johnson, and McKinney (1921) reported from Madison County, Illinois, in that the latter disease exhibits hyperplastic instead of hypoplastic symptoms, there being an excessive production of culms in the Illinois disease; moreover, perithecia are wanting. Take-all differs also from the European foot-rots, the causes of which are usually not given by most investigators, though variously attributed to one or more of the following fungi: *Ophiobolus herpotrichus*, *Leptosphaeria herpotrichoides*, *L. culmifraga*, and *Fusarium* sp. Dr. D. Atanasoff states[5] that the European foot-rot is connected with a pathologic condition of the cereals which has nothing in common with the Australian or the American take-all. The European foot-rots are characterized by a wilting, breaking, and bending-over of fully grown plants, usually single plants or culms that have rotted bases. He says that affected plants are usually uniformly scattered throughout the field. Foex (1919 b) differentiates between the symptoms of take-all and the Leptosphaeria foot-rot, which is more characteristic of the European type of foot-rot, in that the take-all pathogene attacks the base of the stem and the underground parts, causing atrophy and premature dying, while *Leptosphaeria herpotrichoides* causes lodging and produces fruiting bodies higher up the stem.

ETIOLOGY

Nomenclature

The causal organism of the disease is *Ophiobolus cariceti* (B. & Br.) Sacc. In a previous paper (Fitzpatrick, Thomas, and Kirby, 1922) the evidence indicating that *Ophiobolus graminis* Sacc. and *Sphaeria cariceti* B. & Br. are the same organism has been presented.

Pathogenicity

Attempts to prove the pathogenicity of the fungus are reported by Delacroix (1901), Mangin (1902), and McAlpine (1904), each of whom

[5] In a letter to the writer dated March 9, 1923.

inoculated growing wheat with diseased straw or spore suspensions, obtaining typical symptoms of the disease in every case. The first recorded inoculations with the fungus from pure culture were made in New Zealand by Waters (1920, a and b), on wheat seedlings grown from sterilized seed on sterilized soil in test tubes. Inoculated plants died in from twenty-eight to thirty-six days from the date of inoculation, while control plants were in good condition after fifty-eight days. Inoculated culms of wheat plants growing in pots did not become infected.

In the present investigation the relation of *Ophiobolus cariceti* to take-all has been clearly established. The fungus was found regularly associated with the disease in each of the 205 infested fields of winter wheat observed during the four years while this investigation was in progress.

The fungus was isolated by the following methods: Bits of the host tissue bearing one or more perithecia were immersed in a 1-2000 mercuric chloride solution for from one to two minutes and then placed under a binocular, where by means of needles individual perithecia were freed from foreign matter. The perithecia were then crushed in a large drop of sterile water, and this drop was diluted by the addition of sufficient sterile water so that if individual small drops were taken each would contain approximately one ascospore. Drops of this suspension were then atomized on, or several drops were spread over, the surface of dextrose agar[6] in petri dishes. After twenty-four to forty-eight hours, when the petri dishes were examined under the low power of the microscope, the ascospores had started to germinate and single ascospores were transferred with a needle to the surface of agar in petri dishes. During the latter part of the investigation another method[7] was used to obtain cultures from single spores. This consisted in placing individual drops of the ascospore suspension on small pieces of sterile cover slips. The drops on the slips were examined under the microscope, and only those in which the drop contained one ascospore were transferred to the surface of agar in petri dishes. Subsequent examinations were made daily, and where no contamination developed, and the ascospore germinated and produced a colony, a mycelial tip was taken. A third method of isolation consisted in the planting of small bits of diseased host tissue in agar in petri dishes, and the subsequent transfer of single mycelial tips to agar in test tubes.

The first and second methods were used when viable ascospores were obtainable.

Isolation of the fungus from tissue plantings is difficult, because of its slow growth and the occurrence of other more rapidly growing organisms in the tissue. Nevertheless the fungus was isolated from fifty tissue plantings, and more than one hundred and ninety ascospore dilution

[6] Formula; 100 cubic centimeters water, 20 grams agar, 2 grams dextrose.
[7] This method, as well as the formula for the agar, was suggested to the writer by Dr. H. E. Thomas.

cultures were made, in the comparative study of the fungus in pure culture from many sources. The growth of this fungus on various media is in general of two types, examples of which are as follows:

Type I: On 0.2-per-cent dextrose agar the growth (Plate III, A and B) reaches 2.5 centimeters in diameter after seven days, and seldom exceeds from 5 to 7 centimeters in thirty days or longer. The mycelium after seven days of growth is submerged or wholly appressed. The colony is translucent and is made up of feathery, rhizomorphic strands resembling the feathery branchings of some mosses. After thirty days the appearance of the colony is nearly the same as at the end of seven days, except that it has become in color a deep neutral gray[8] and hyphal strands have appeared near the center. The rhizomorphic strands are appressed to the surface or submerged. Single hyphae radiate toward the edge of the culture. The cells of the hyphae are from 16 to 45 μ long and from 4 to 5.5 μ in diameter, and contain from 10 to 25 guttulae. The cell walls are hyaline. Branches arise near the tip of about every eighth to tenth cell, or at a distance apart of about every 150 to 250 μ. The cells of the branches are much more variable in size and shape than those of the main hypha, ranging from knob-like cells with a diameter of 5μ, such as occur at the end of a few hyphae, to cylindrical cells resembling those of the main hypha.

Type II: On potato agar (5.4 pH) the growth (Plate III, E and C) reaches 7 centimeters in diameter at the end of seven days, and covers the plate (9 centimeters) in from eight to nine days. After seven days of growth the mycelium is hyaline, and is submerged or wholly appressed. The individual colony is abundantly and distinctly flexuous; its margin is even but is not marked by a definite line, appearing finely frayed. At thirty days the mycelium of the colony has assumed a more pronounced aerial habit, rising to a height of 2 millimeters and growing on the sides of the petri dish. The aerial growth causes the surface to appear as if covered with a silvery bloom. The colony ranges in color from a neutral gray to a deep neutral gray. The surface of the medium is thickly covered with tortuous, mixed, ribbon-like, rhizomorphic bands of hyphae. These bands are made up of from two to eight strands of parallel hyphae. The cells of the individual strands are cylindric, and have thick, dark neutral gray walls; they are 75 to 260 μ long and from 4 to 6.5 μ in diameter (averaging 184 by 5 μ). The contents are hyaline and sparingly guttulate. Nearly every cell in the strands sends forth a branch at its tip. The cells of the hyphal strands closely resemble those of the plate mycelium on the host (Plate III, C). The branches terminate in thin-walled hyaline hyphae, the individual cells of which contain from 10 to 30 guttulae. The cells are very variable in shape and size, ranging from 15 to 50 μ long and from 2 to 6 μ in diameter. Anastomosing is observed frequently.

[8] Ridgway, Color standards and nomenclature.

Type I, which is characterized by the radiate habit of the strands, occurs only on the 0.2-per-cent dextrose agar. Type II, which is characterized by the strands intermixing on the plate and forming ribbon-like strands of parallel hyphae, occurs when the fungus grows on the following agar media: potato, cornmeal, bean, oatmeal, prune, nutrient, Czapek, crushed wheat (Kirby, 1922), and 2- and 5-per-cent dextrose. On crushed wheat agar aerial growth is much more abundant than on any of the other media. Zonation does not occur on potato agar with a pH of 5 to 6, but it does occur and becomes increasingly pronounced as the pH value decreases or increases.

The writer has been able to develop perithecia in culture, both in petri dishes containing crushed-wheat agar and on sterilized sweet clover and wheat stems. Different monosporous isolations of the organism showed a marked difference in their capacity to develop perithecia. Some of these were greatly stimulated in their development of perithecia by being grown in apposition with other monosporous cultures. From a considerable number of these isolations perithecia were not obtained except when they were grown in this manner. However, this was not true of all the cultures studied, since some of them were found to produce perithecia without stimulus of a second strain. It was believed in the earlier part of the work that the majority of the strains of the fungus exhibited a true heterothallism (Kirby, 1923). Subsequently strains have been found which are not identical in their cultural reactions with either of the original plus or minus types. Further investigation will be necessary to determine whether or not the strains of this fungus can be segregated into definite sex types.

The characteristic signs and symptoms of the disease have been produced by inoculations of pure cultures of the fungus into healthy plants. In several series of inoculation experiments, the soil of 457 pots contaminated with pure cultures of the fungus at the time of planting were variously sown with wheat, rye, barley, or other species of grasses. Within four months after planting, characteristic symptoms and signs appeared on plants in all the pots, while the plants in 212 pots planted under the same conditions excepting that no inoculum was placed in them never showed signs or symptoms of take-all.

In a field which was known to be free of take-all, 64 rod-rows of winter wheat of different varieties were inoculated in the fall of 1921 with pure cultures of the fungus, and in every row the same characteristic signs and symptoms of the disease were produced as have been found in all infected fields observed in New York. In this test field, the same symptoms and signs appeared when 47 rod-rows of winter wheat were inoculated in the fall of 1921 by placing diseased straw brought from other fields with the seed at planting time. Further take-all symptoms and signs appeared when 45 rod-rows of winter wheat were inoculated with pure

culture and diseased straw in the fall of 1922. In the same tests, 209 rod-rows of winter wheat growing more than one foot distant from inoculated rows, which received no inoculum, produced no signs or symptoms of take-all. Typical symptoms of the disease were produced when the roots of fifty sterile wheat seedlings were inoculated with a pure culture of the organism growing in large individual test tubes. At the same time, twenty uninoculated seedlings growing under the same conditions showed no symptoms of the disease.

The fungus was reisolated at least twelve times from the lesion areas on plants that had been inoculated with pure cultures, but it was never isolated from bits of the host tissue taken adjacent to the discolored area. The reisolated fungus agreed in every essential with that in the original cultures. Furthermore, when used as inoculum at planting time, these cultures gave typical symptoms of take-all on the growing wheat plants in the field. There can be, therefore, no doubt that the fungus *Ophiobolus cariceti* is the cause of the take-all disease.

Life history

The life history of this fungus exhibits no definite separation into primary and secondary cycles. Infection appears to take place at any time during the year when moisture and temperature conditions are favorable. It appears to be initiated usually by ascospores, but there is evidence that the fungus may spread from plant to plant by mycelium growing through the soil.

Pathogenesis

Inoculation.— The chief sources of inoculum are the lower internodes and the roots of diseased plants. Upon these perithecia are formed during a period of four weeks preceding harvest. The rootstocks of *Agropyron repens* have been observed to harbor mycelium of the take-all pathogene for twelve months. Wheat planted among these rootstocks soon showed signs and symptoms of the disease. Thus mycelium-harboring grasses are another important source of inoculum. Winter wheat planted in September in soil removed from infested wheat fields the previous spring has, in both 1920 and 1921, become infected. In this case mycelium harbored in the soil during the summer probably acted as the inoculum in the fall. Concerning the source of this inoculum, Spafford (1917) states that it appears to come wholly from the soil, particularly from straw of affected plants left in the field.

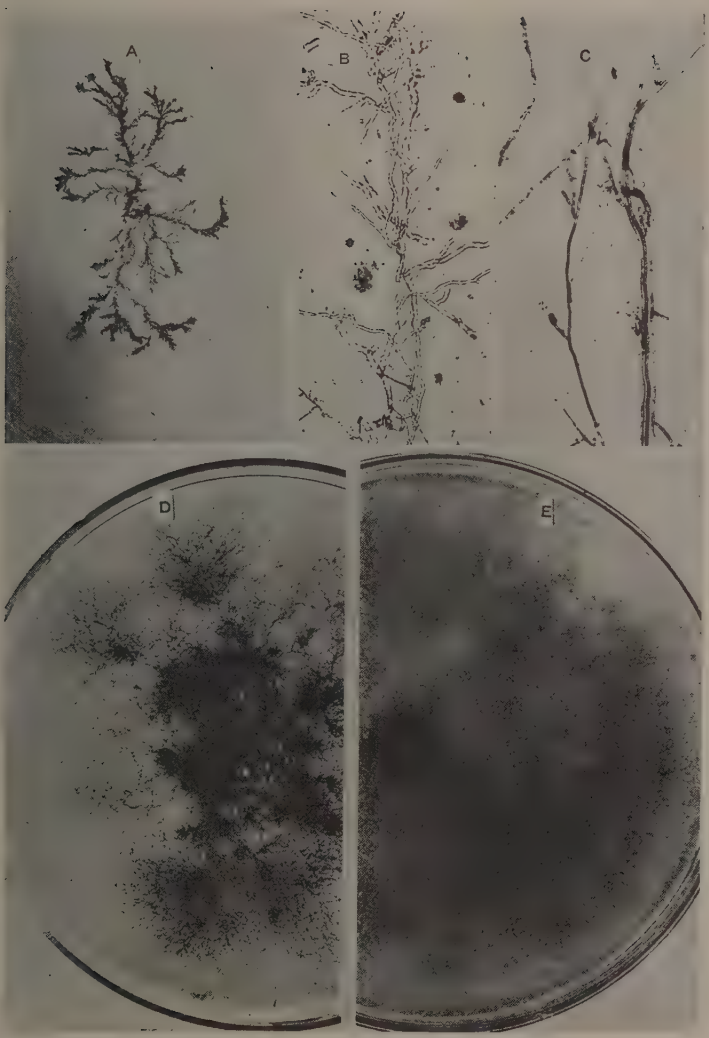
Field observations have indicated that the take-all fungus may be harbored with the seed to a very small extent. However, repeated plantings of seed from diseased plants in the greenhouse have given negative results. In an effort to ascertain whether the pathogene is transmitted with the seed, the following test was made in the fall of 1921 on land

HEALTHY AND TAKE-ALL-AFFECTED WHEAT PLANTS

A, (left) Healthy wheat heads, (right) take-all-affected wheat heads. B, (left) Healthy plant with five heads, height 40 inches; (right) diseased plant with one shriveled head, height 18 inches. C, Plants after four months growth: pot 13, check; pots 11 and 12, diseased straw mixed with soil at planting time

SYMPTOMATOLOGY OF THE DISEASE, AND MORPHOLOGY OF THE PATHOGENE

A, Base of wheat plant, showing lesions on culms and on roots. B, Asci and ascospores (× 130.3). C, Longitudinal section of a wheat plant showing structure and contents of the perithecia (× 39.4). D, Two perithecia developed below the outer leaf sheath; the oblique beaks illustrate one of the most characteristic features of the species (× 24). E, The lower part of a culm of a wheat plant infected with take-all, showing protruding beaks of the perithecia (× 3.3). F, A part of the mycelial plate found about the base of the culm (× 130.3)



THIRTY-DAYS-OLD CULTURE OF OPHIOBOLUS CARICETI ON 0.2-PER-CENT DEXTROSE AGAR AND ON POTATO AGAR

A, Culture on 0.2-per-cent dextrose agar (× 8.9). B, Characteristic rhizomorphic strands of mycelium as found on 0.2-per-cent dextrose agar (× 450.7). C, Characteristic ribbon-like rhizomorphic bands of mycelium as found on potato agar (× 450.7). D, Culture on 0.2-per-cent dextrose agar. E, Culture on potato agar

which was known to be free from the organism. Twenty-one rod-rows of winter wheat were planted with seed obtained by hand-threshing diseased plants collected from all the infested fields surveyed in 1921. Special care was taken in the threshing to see that no part of the culms became mixed with the seed. No symptoms nor signs of take-all were observed in any of these rows. In addition to this test, 77 rod-rows of winter wheat were planted with seed that came from a badly diseased field, the grain of which was threshed by the usual methods. One or two diseased plants appeared at one place in these rows. This seemed to indicate that the pathogene may be carried with seed in which bits of diseased culms have become mixed.

It appears, however, that the ascospores constitute the inoculum for the vast majority of infections. The perithecia in which they are developed are usually produced in great numbers on wheat, on barley, and on many grasses. These minute dark brown to black, rostrate fruit bodies (Plate II, C and D) are most commonly and abundantly developed beneath the leaf sheaths of the lower internodes. They occur also on the roots in the thick wefts of fine rootlets developed above the diseased crown. They are gregarious or scattered. When young the perithecia are hidden, but at maturity their prominent beaks protrude through the leaf sheath.

These perithecia contain numerous elongate clavate asci, each containing eight spores (Plate 11, B). These ascospores are long, slender, and hyaline, and are continuous when young but at maturity are 5–7–septate. They measure 60 to 107 by 3 to 3.75 μ. They do not usually become fully mature until early autumn.[9]

In the examination of several thousand individuals it has been noted that soon after formation the perithecium contains a few apparently mature and many immature ascospores, and that during the following nine months it always contains a proportion of immature ascospores. Hori (1901) states that in Japan the ascospores germinate in June and July. McAlpine (1904) reports that in Australia they germinate during November, and Robinson (1907) finds them capable of germinating immediately after formation if sufficient moisture and air are present. However, no investigator has reported on a careful study of their longevity.

With a view of ascertaining the length of the period during which ascospores will germinate, field material was collected and stored under field conditions in 1920, 1921, and 1922. During those three years attempts were made to germinate the ascospores nearly every month. The results of these tests show that ascospores formed in June and July have in some years failed to germinate and have in other years germinated to a small extent (less than 5 per cent) in July and August of the

[9] A detailed description of the morphology of the perithecium has been given by Fitzpatrick, Thomas, and Kirby (1922:36–37).

same year. In September the germination increased to about 25 per cent, and the maximum (50 per cent) was reached during October, November, and December. The percentage of germination fell off gradually during January, February, and March. The latest germination to be observed was in March and April, after which all the ascospores failed to germinate. Thus it appears that most of the ascospores come to maturity and are discharged soon after the winter wheat is planted in the autumn, at which time inoculation and infection usually occur.

The manner of ascospore discharge from the perithecium has been studied by Hori (1901), McAlpine (1904), and Waters (1920 a). These investigators all agree that the ascospores escape through the ostiole but that they are not shot forth into the air. McAlpine stated further that the asci are expelled one after another, and the ascospores are then set free in the presence of moisture. Waters (1920 a) pictures perithecia which have pushed out coils of mucilaginous matter carrying the asci through the neck with it. He states that this takes place in slightly moist air, that in very moist air a globose mass is formed at the ostiole, and that in water the moisture dissolves the mucilage and liberates the spores as the asci emerge from the ostiole. A dry mass of asci and ascospores has several times been observed by the writer to have formed at the ostiole, where it remained for several months if there was no rain to dissolve it. Apparently asci and ascospores are discharged only when there is sufficient moisture present to soak up the contents of the perithecium. Perithecia formed in pure culture have not been observed to liberate their spores until placed in water. Field observations seem to indicate that asci and ascospores are discharged during rainy periods, and that usually the splashing rain removes them before they can accumulate at the ostiole.

The date at which ascospores are mature and ready to be discharged may be determined by immersing perithecia from time to time in water, when, if mature, the ascospores will be exuded. Hori (1901) states that in Japan at least some spores are discharged within ten days after the crop is cut, or about June 23, and Waters (1920 a) states that in New Zealand perithecia put forth tendrils of spores in January (corresponding to July in New York). From observations made by the writer, it seems that the date of discharge is greatly influenced by the time of formation of the perithecia and the subsequent moisture conditions. Perithecia have developed as early as June 4 and have been observed discharging their ascospores within two weeks after their formation. It has been noted that perithecia seldom discharge all their ascospores, but retain a certain number which finally disintegrate during April and May following their formation. Ascospore discharge may begin as early as the latter part of June, but it probably does not reach its maximum until September and November, ceasing some time in March or April of the following spring.

It is clear that the behavior of the ascospores at the time of their discharge restricts the agents of dissemination almost completely to rain. To determine whether they could live long enough to be wind-borne, the following test was made. A suspension of ascospores and asci was prepared, small drops of which were placed on cover slips and allowed to dry. At definite time intervals after the drops had dried, water was again added and the cover slide was put over a Van Tiegem cell in an attem pt to germinate the spores. The results were as follows: ascospores in unexposed controls germinated 49 per cent; free ascospores exposed for two minutes germinated 2 per cent; free ascospores exposed for three, five, ten, and thirty minutes, and one, two, and twelve hours, failed to germinate. Asci exposed for three minutes showed ascospores germinating in 40 per cent; those exposed for five minutes showed 35 per cent germination; those exposed for ten minutes showed 16.6 per cent germination; and those exposed for thirty minutes and for one, two, and twelve hours failed to show any germinating ascospores.

Hori (1901) states that spores are disseminated in the soil near the host and that wind may carry them for a short distance. Results of the present investigation indicate that ascospores, and to a certain extent asci, are so susceptible to drying that they cannot be transported in this manner. Besides the dissemination of single ascospores, the fungus may be carried as perithecia on host tissue. Waters (1920 a) states that infected fragments may be carried by animals, wind, and cultivating tools, and McAlpine (1904) believes that much of the dissemination of the fungus in parts of Australia is due to dust or wind-blown soil. Mycelium harbored on the rootstocks of perennial grasses may be scattered by cultivation.

Mangin (1899), Hori (1901), McAlpine (1904), and Waters (1920 a) have all stated that the roots serve as the infection court and that they were unable to get infection on the upper part of the wheat plants. The results obtained during the present investigation corroborate their findings.

Incubation.— In water cultures, ascospores germinate either when free or while still in the ascus. Germination begins in about ten hours, by the pushing-out of a germ tube from one or both ends and in some instances at points near the middle of the ascospore. A knob-like swelling, of a diameter of 5 to 8 μ, has been observed to develop at the end or along the middle of the ascospore. One or more germ tubes emerge from the distal end and the sides of this swelling.

Sickle-shaped microconidia, which seem to be functionless, may bud out from the ends and the sides of the ascospores. These microconidia are hyaline, measuring 5.5 to 8 by 1.5 to 2 μ. They are borne also on the ends of certain hyphae which function as conidiophores. Foex (1919 c) considered them sporidia and called them falciformers. McAlpine (1904) recorded their presence without giving any hint as to their function.

The germ tube, after its first appearance, grows rapidly and may reach a length of 55 μ and 300 μ in 24 and 48 hours, respectively. The germ tube is hyaline. Septa are not developed until during the second day's growth. Guttulae or fat globules are found in the germ tubes after the septa are laid down. These observations agree with those of Delacroix (1901), McAlpine (1904), and Hori (1901), except that Hori appears never to have observed germ tubes arising from the middle of the ascospore.

The effect of varying degrees of acidity on ascospore germination was determined by germinating ascospores in thirteen different solutions prepared with hydrochloric acid and sodium hydroxide and ranging in acidity from pH 3 to 10. The results of this test are shown in figure 2. The bimodal germination curve shows that ascospores of this fungus

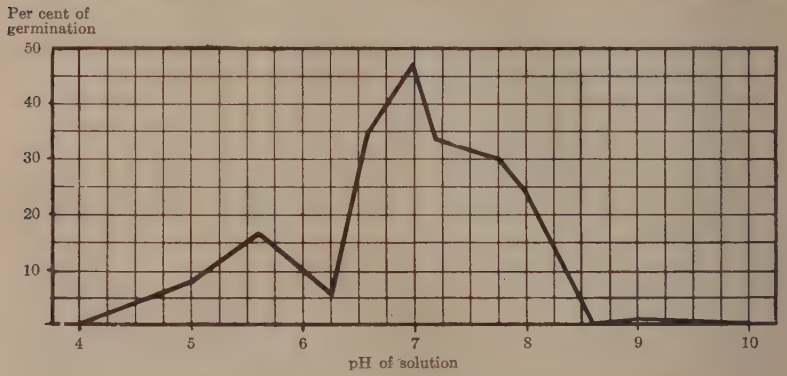


FIGURE 2. PERCENTAGE OF ASCOSPORES OF OPHIOBOLUS CARICETI GERMINATING AT VARYING DEGREES OF ACIDITY

germinate through a smaller range of acidity than that in which growth takes place. The rate of growth of the germ tubes in the solutions of different pH values was greatest at pH 7 and was only slightly less at pH 8 (hyphae 180 μ long in 48 hours .

Concerning penetration, Mangin (1899) reports that the germ tube creeps along the surface of the root hairs and soon pierces the membrane and enters the interior of the plant. In the present investigations, wheat plants growing in pure culture in test tubes on 0.2-per-cent dextrose and potato agars in petri dishes were inoculated with ascospores, and the growth and penetration of the germ tube were observed under the microscope. Approximately fifty inoculations were made in this manner, and it was noted that the germ tube of the pathogene grew along the surface of the roots and nearly always entered the epidermis directly. In one case a hypha formed a small appressorium on a root hair, and from this

a penetration tube entered and passed down the root hair into the root; but in the other four cases of root-hair penetration observed, appressoria were not formed.

Infection.— In wheat plants growing on agar plates the first evidence of infection appeared seven days after the fungus had come in contact with the roots, and consisted of small, brown, necrotic spots. These spots enlarged so rapidly that within a period of from three to five days the entire root for a distance of one-quarter to one-half inch was dead and brown, all of the tissues being permeated with mycelium. Robinson (1907) states that in Australia young seedlings planted in diseased soil may be affected almost as soon as they appear above ground. McKinney (1922) reports that plants in the greenhouse show severe seedling blight and often die within fourteen days after sowing.

Under New York field conditions, symptoms of the disease have not been observed on the culms before May 20, but lesions have been found on the roots as early as March 19. The fungus has been isolated from such lesions.

The death of the roots reduces the supply of moisture and mineral nutrients to the plants. To overcome this, many fine rootlets are developed above the lesion. The plant may also send out new roots at its crown. These efforts of the plant to recover seem to be of little value when it is attacked while young, since the mycelium grows upward inside and outside of the roots to the crown, attacking and killing all roots with which it comes in contact. The mycelium enters the leaf sheaths at the crown, permeating the tissues.

The septate mycelium is of two general types, colorless and brown. The very young mycelium, and nearly all the mycelium within the culm and the roots, is colorless. This consists of rhizomorphic strands, the individual cells of which are cylindric, thin-walled, hyaline, and 10–30-guttulate. These cells are from 15 to 50 μ long and from 2 to 6 μ wide. The cells of the branches are very variable, ranging from knob-like cells with a diameter of 5 μ, to cells resembling the main rhizomorphs. The mycelium is abundant in the xylem tubes and grows intracellularly in all the other tissues.

By the time the mycelium has entered the leaf sheaths, the roots are usually so severely affected that the plant becomes yellow. Soon after this symptom appears, a mass of mycelium is formed between the inner leaf sheath and the culm. This mass is composed of hyphae which intermingle to form a plate, which usually adheres to the culm when the leaf sheaths are stripped away. The plate mycelium thus formed consists of flat, ribbon-like strands resembling somewhat compressed rhizomorphs, which run definitely parallel to one another. The individual cells of these strands are cylindric, and are from 20 to 260 μ long and from 3 to 6.5 μ wide. Their contents are sparingly guttulate and hyaline.

Saprogenesis

With the formation of the plate mycelium the plant usually succumbs, and thereafter the fungus leads a saprophytic existence. Perithecia are now formed on the inner side of the outer leaf sheath and in mycelial mats covering the roots. These leaf sheaths and roots are nearly always dead before perithecia are developed, and often the entire plant has died before the perithecia reach their full size. Fully developed perithecia have been formed on wheat in the greenhouse within eight weeks after planting of the seed, but in the field they have not been found before June 4. The ascopores which are discharged early in the summer are probably washed to the ground, where they germinate and produce mycelium which may live saprophytically in the soil on organic matter until the fall, when wheat is planted. The mycelium in the soil about the cereal or grass plants may also live saprophytically until fall.

The ascospores in the perithecia are only partly discharged at any one time, as already pointed out. Hori (1901) states that the ascospores may germinate and live saprophytically on the decayed culm until autumn, when the mycelium attacks the young cereal plants.

While the life cycle of this pathogene is usually initiated by ascospores, mycelium spreading through the soil also may infect healthy plants. Hori (1901), McAlpine (1904), Waters (1920 a), and others have noted that the infection occurs and seems to spread in circular areas. Spafford (1917) states that mycelium spreads from root to root by dark brown mycelial strands. During the present investigations it was noted that in infested wheat fields the margin of the spots killed by take-all was composed of a border, ranging in width from 6 inches to several feet, wherein the infected plants were not stunted and seemingly had been attacked late in their development. The 1921–22 plot gave ample opportunity for observing the spread of the pathogene through the soil, since inoculum was placed only at known points in the field. In these plots it was clearly shown that during the period of growth of winter wheat the mycelium could not have spread through the soil for a distance greater than one foot. Only a fraction of one per cent of the plants were killed in their later stages of growth at this distance from inoculum, consisting of pure cultures or diseased wheat straw, placed in the ground at planting time. Thus it seems that the fungus is able to spread through the soil for very short distances only and initiate new life cycles.

ECOLOGY

Moisture and temperature

There seems to be a direct correlation between such climatic factors as moisture and temperature, and the amount and severity of take-all. It is said (Anonymous, 1922 b) that climatic factors have a tremendous

influence on the severity of the disease in Oregon, and that it appears probable that only in certain especially favorable seasons will the disease result in serious crop damage. Guerrapain and Demolon (1913) conclude that high autumn temperatures promote the infection of seedlings in France. Dombrovski (1909) and Mangin (1914) state that high humidity of the soil favors the disease. Robinson (1907) believes that a dry summer followed by a wet winter affords the most favorable conditions for fall infection. Brittlebank (1920) reports that water-logging of the soil before harvest, with a subsequent drying, favors the development of the disease.

Observations made during the present investigations seem to corroborate in general the foregoing statements regarding the effect of climatic factors. In most of the infested wheat fields examined there was little apparent difference in the amount of infection on high and low ground, although in a few fields the infection appeared to be much heavier on the lower and wetter soils. In the 1922 test plot an inoculated check row in water-logged soil had a higher percentage of badly diseased plants than did the rows on drier soil.

Varying climatic conditions which occurred during the springs of 1921, 1922, and 1923 brought about marked differences in the amount and severity of take-all. As shown in figure 3, the spring of 1921 was warm and wet during March and April, while May and June were exceptionally dry. Under these conditions there occurred a higher percentage of extreme stunting, combined with a premature dying of the infected winter-wheat plants.

The spring of 1922 differed from that of 1921 in that it was cooler or rather later, and in May, and especially in June, there was greater rainfall. Under these conditions about 40 per cent less plants were found to be diseased in the infested fields than in 1921. The plants that were affected died later and the amount of stunting was much less.

The spring of 1923 was abnormally late, cool, and dry, with the low rainfall continuing until harvest time. Under these conditions the disease was far less severe than in either of the two preceding years. This decrease in the severity of the disease is shown by the fact that in 1923 the disease appeared two weeks later than ever before, and 70 per cent less plants were found diseased in 1923 than in 1922; also, in 1923 there was less dwarfing of the affected plants than in any previous year.

Thus the weather conditions which seem to favor the disease are abnormally high temperature and rainfall in the early spring (March and April), followed by dry, hot weather from the period of jointing until harvest. Springs in which abnormally high temperature and rainfall occur seem to be less conducive to the development of the disease if during May and June the rainfall is abnormally high and the temperature is normal or below.

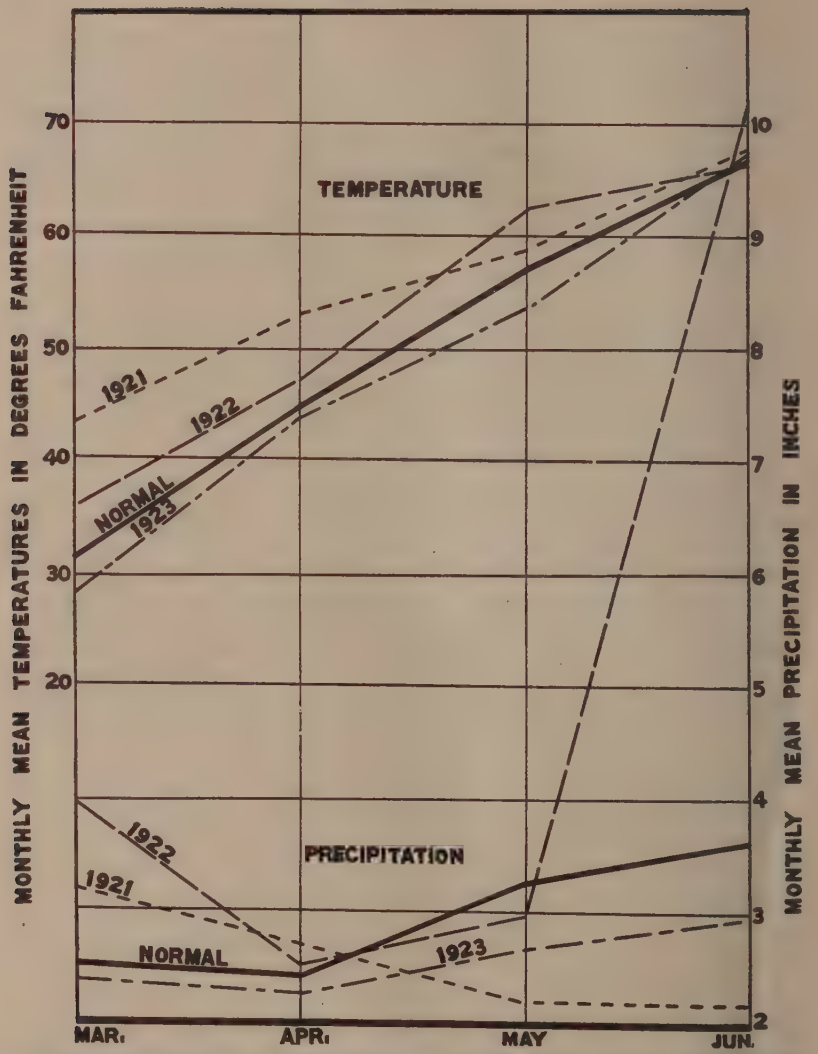


FIGURE 3. THE MONTHLY MEAN TEMPERATURES AND PRECIPITATION FOR FOUR SPRING MONTHS IN 1921, 1922, AND 1923, COMPARED WITH NORMAL TEMPERATURE AND PRECIPITATION
The mean in this data is the average of conditions at Ithaca, Syracuse, and Rochester

Abnormally low temperatures and rainfall in the early spring, such as occurred in 1923, seem to be very unfavorable to the development of the disease.

McKinney (1922) reports that the most favorable temperature for the fungus to attack wheat seedlings is near 22° to 24° C. This explains why take-all may be more destructive in the earlier-planted winter wheat. After the time of early wheat planting the temperature is often above 22° C., but after the time of the late planting the temperature seldom reaches this point.

Soil fertility and acidity

Several investigators have studied the relationship between the type of soil and the severity of take-all. Hartmann (1914) and Gray (1914) state that the disease is worse on sandy, light soils than on heavy, stiff, clay soils; while Hori (1901) believes that in Japan the disease is worse on clay or loamy clay than on sandy soils. Richardson (1911) states that take-all occurs on all types of soil.

Many workers have investigated the influence of various fertilizers and other substances on the development of the disease. Rosen and Elliott (1923) found that under Arkansas conditions commercial fertilizer almost completely prevented the disease while manure decreased the percentage of diseased plants to a considerable degree. Hori (1901), working in Japan, found that the intensity of the damage apparently bears a close relationship to the nature and amount of the fertilizer applied to the soil. The greatest damage occurred when an excessive amount of nitrogenous fertilizer was applied or when the three essential elements were unbalanced. Lime has generally been reported as greatly increasing the amount of disease (Hartmann, 1914, Brittlebank, 1920, and Rosen and Elliott, 1923). Superphosphate of lime has been reported as increasing (Brittlebank, 1920), as not checking (Mangin, 1902), and as decreasing (Anonymous, 1913) the growth of the fungus. Copper sulfate in some cases had no particular effect in the control of the pathogene (Foex, 1919 b), while in other cases it caused a reduction in the amount of the disease (Darnell-Smith, 1916, Foex 1919 b and Benoist and Bailly, 1922). Sulfuric acid and chloride of potassium resulted in a marked reduction in foot rot caused by *Ophiobolus cariceti* and *Leptosphaeria herpotrichoides* (Benoist and Bailly, 1922).

From these more or less conflicting reports there appear to be two factors which decrease the amount of take-all: first, the type of soil fertility which enables the plants to grow at a moderate rate, but not with a profuse vegetative growth such as occurs when an excessive amount of nitrogen is present; and secondly, an acid condition of the soil. To test the fertility factor in a preliminary way, the following experiment was run in the greenhouse. Twenty-five 5-inch pots were filled with clean soil and were inoculated at the same time and under the same conditions

as those of the first part of the host-range test, except that five kernels of No. 6 Junior wheat were planted in each pot. The twenty-five pots were divided into five series of five pots each. The first series of pots received no fertilizer, while each of the other four series received one of the following treatments:

1. One gram of sodium nitrate per pot, applied by dissolving in 100 cubic centimeters of water which was then poured over the soil.
2. One-half gram of acid potassium phosphate (dibasic) per pot, mixed with the soil.
3. One gram of sulfur per pot, mixed with the soil.
4. Three grams of hydrated lime per pot, mixed with the soil.

After seventeen weeks of growth, no marked differences were found among the different series. Later, the series to which lime had been added was the first on which perithecia were found. This series had more killed plants (15.7 per cent) than the sodium nitrate series (13.6 per cent), the acid potassium phosphate series (5 per cent), or the sulfur series (none), and had less dead plants than the series to which no fertilizer had been added (25 per cent).

After eight months of growth, the plants in all of the series except the one to which sulfur had been added were heavily infected, were badly stunted, and had produced no heads. Even the plants in the sulfur series showed considerable infection, but those in three of the pots had produced several heads. The sulfur apparently produced an acidity of the soil which seemed to have delayed the attack of the fungus or lessened its severity.

From the literature and the preliminary test on the effect of fertilizers, there appears to be a definite relation between the hydrogen-ion concentration in the soil, and the severity of take-all. In order to further test this point, the fungus was grown in culture on an acidity series consisting of potato agars of nineteen different degrees of acidity ranging from pH 3.2 to 9.6, and on bacto-cornmeal agars of ten different degrees of acidity ranging from pH 3 to 9.2. The degree of acidity of the media was determined by the colorimetric method as recommended by Clark.[10]

Cultures of *Ophiobolus cariceti* from three sources, in comparison with one strain of *Fusarium moniliforme* Sheld., were grown on the media in total darkness at room temperature (15° to 20° C.). The average growth of the three strains of *Ophiobolus cariceti*, and the growth of the single strain of *Fusarium moniliforme* at the end of eight days, are shown in figure 4. The results indicate that the take-all fungus requires a condition of alkalinity for optimum growth, and may explain why additions of alkaline substances to the soil have been reported as favoring the fungus while additions of acid-forming substances decrease the amount of infection.

[10] Clark, W. Mansfield. The determination of hydrogen ions, p. 1–317. 1920.

In order to determine the effect of acidity on the amount of infection, the following test was made in the greenhouse. Sodium hydroxide and sulfuric acid were added in varying amounts to lots of soil so that eighteen degrees of acidity, ranging from pH 2.3 to 8.8, were obtained. The soil comprising each degree of acidity was distributed in five 5-inch pots on February 17, 1922, and five seeds of No. 6 Junior wheat were planted in

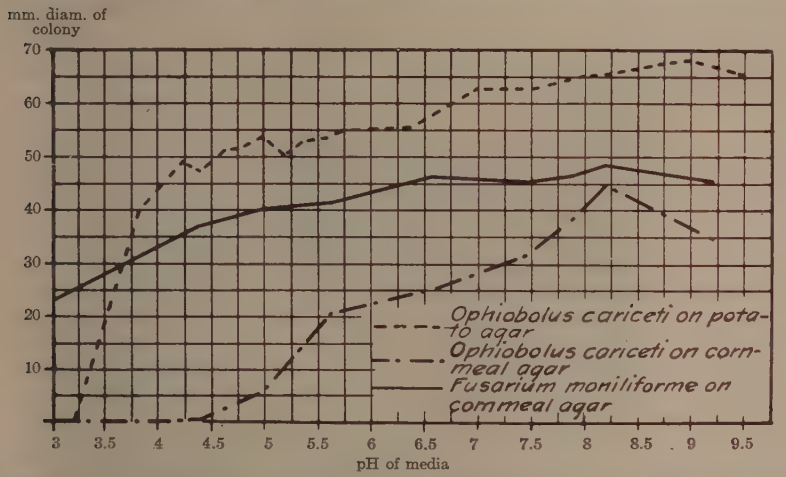


FIGURE 4. RATE OF GROWTH OF OPHIOBOLUS CARICETI AND FUSARIUM MONILIFORME ON AGARS HAVING VARYING DEGREES OF ACIDITY

each pot. In three of the five pots a pure culture of *Ophiobolus cariceti* was added at the time of planting. Within three months after the date of planting, the plants growing in inoculated soils having a pH value from 5.2 to 8.6 were all dead. The plants growing in soils having pH values of 3, 4, and 2.3 were still alive and showed few symptoms of take-all. These plants remained alive until the test was discontinued at the end of nine months. They never grew higher than a few inches, because of the unfavorable soil condition. The plants in the soil having a pH of 8.8 remained alive for about six months, when they all succumbed to the attack of the disease. This experiment seems to show that when sufficient inoculum is present and the plants are grown under greenhouse conditions, no degree of acidity at which plants will normally grow to maturity will allow them to escape being killed by the disease. The plants in the uninoculated pots produced the most heads and grew the tallest at pH values between 7.8 and 8.4, and no heads were produced by plants growing in soil having a pH value as low as 3.4.

In order to determine further whether acidity of the soil was an influencing factor on the disease, a test plot at Ithaca was laid out in which certain substances were applied to the soil. There were six series of eleven rod-rows each, which differed from one another only in the substance added. In the first series lime was applied at the rate of 1500 pounds per acre; in the second, sulfuric acid, 300 pounds per acre; in the third, lime, 500 pounds per acre; in the fourth, sulfuric acid, 100 pounds per acre; in the fifth, sulfur, 1500 pounds per acre; and in the sixth, sulfuric acid, 50 pounds per acre. The arrangement of the rod-rows in each series for testing each of the above substances was as follows:

Row 1	No inoculum	No substance added
Row 2	Pure culture of *Ophiobolus cariceti*	No substance added
Row 3	Pure culture of *Ophiobolus cariceti*	No substance added
Row 4	Pure culture of *Ophiobolus cariceti*	No substance added
Row 5	Pure culture of *Ophiobolus cariceti*	No substance added
Row 6	Pure culture of *Ophiobolus cariceti*	Substance added
Row 7	Pure culture of *Ophiobolus cariceti*	Substance added
Row 8	Pure culture of *Ophiobolus cariceti*	Substance added
Row 9	No inoculum	Substance added
Row 10	No inoculum	Substance added
Row 11	No inoculum	Substance added

All rows were planted with No. 6 Junior wheat, and the inoculum and acidifying substances were added on the day of planting. During July, 1922, the rows were harvested by digging up the plants and taking the percentage of diseased plants, the degree of stunting, and the yields. These data, summarized in table 4, show that the addition of lime increased the yield of take-all-free plants, but that it increased the percentage of infected and badly stunted plants to such an extent that the rows yielded less than did those that received an application of acid. This was due to the fact that the healthy plants in the rows growing in the very acid soil yielded only half as much as did those in the very alkaline conditions.

Date of planting

Hori (1901) states that in Japan there is a close relationship between the time of sowing and the severity of take-all, and cites several cases in which barley sown between October 13 and October 27 was much more heavily damaged than was that sown two or three weeks later. In order to get data on the relation of the date of sowing to the amount of disease, two rows each of No. 6 Junior and Forward wheats were sown in infested soil on September 6 and September 29, 1921. The results which are given in table 5, show that later planting decreases the percentage of disease, but at the same time it decreases also the number of heads per plant.

TABLE 4. EFFECT ON THE AMOUNT AND DEGREE OF SEVERITY OF TAKE-ALL, OF ADDING ACID- AND ALKALI-FORMING SUBSTANCES TO THE SOIL

Substances added to soil at planting time	Weight of seed produced by 100 plants* (grams)	Plants infected with *Ophiobolus caricetî*: Stunted less than ½ normal height: Per cent of plants	Number of culms per plant	Weight of 10 heads (grams)	Average weight of seed per plant (grams)	Stunted ½ to 9/10 normal height: Per cent of plants	Number of culms per plant	Weight of 10 heads (grams)	Average weight of seed per plants (grams)	Little or no stunting: Per cent of plants	Number of culms per plant	Weight of 10 heads (grams)	Average weight of seed per plant (grams)	Plants not diseased: Per cent of plants	Number of culms per plant	Weight of 10 heads (grams)	Average weight of seed per plant (grams)
1500 pounds of lime per acre	38.88	47.0	0.14	0.065	0.0009	41.0	1.12	2.845	0.319	12.0	2.7	6.4	1.73	0.0	....	.....	.....
500 pounds of lime per acre	90.57	12 0	0.33	0.35	0.0011	45.0	1.09	2 845	0.312	37.0	1.68	9.54	1.58	6.0	2.6	11.6	3.01
No application	79.46	30.0	0.50	0.30	0.015	33.5	1.20	2.82	0.348	33.5	2.1	8.3	1.77	3.0	2.5	11.4	2.85
50 pounds of sulfuric acid per acre....	72.78	26.0	0.61	0.942	0.0574	34.0	1.40	2.81	0.394	37.0	1.7	8.25	1.40	3.0	2.4	9.916	2.38
100 pounds of sulfuric acid per acre....	127.95	8.5	1.00	1.02	0.102	13.5	1.7	2.902	0.495	63.0	1.8	7.932	1.43	15.0	2.2	9.19	2.02
300 pounds of sulfuric acid per acre....	129.50	4.5	1.00	1.23	0.123	25.0	1.36	3.59	0.489	47.5	1.72	8.78	1.51	23.0	2.5	7.812	1.957
1500 pounds of sulfur per acre.....	111.56	0.0	....	.....	.....	4.0	2.00	2.72	0.54	31.0	1.07	6.34	0.676	65.0	1.87	7.92	1.36

* The figures in this column represent the sums of the weights of 100 plants taken from the respective plots in the proportions indicated by the percentage of plants in each of the four classes, that is, (1) those less than one-half the normal height, (2) those from one-half to nine-tenths of the normal height, (3) those showing little or no stunting, and (4) those not diseased.

TABLE 5. RELATION OF DATE OF SOWING TO AMOUNT OF DISEASE

Date planted	Take-all-infected plants						Plants not infected	
	Plants less than ½ normal height		Plants ½ to $\frac{9}{10}$ normal height		Plants of normal height			
	Per cent of plants	Number of heads per plant	Per cent of plants	Number of heads per plant	Per cent of plants	Number of heads per plant	Per cent of plants	Number of heads per plant
September 6	9.0	0.43	15.0	2.2	70.0	2.5	6.0	2.4
September 29	2.0	0.48	14.8	1.4	70.6	1.1	12.6	1.1

Rate of seeding

In Europe, where take-all is not sharply defined from several other foot rots, Guerrapain and Demolon (1913), Mangin (1914), and Foex (1919 b) all state that increasing the rate of seeding is favorable to the disease. In order to determine this point, test rows of winter wheat were planted in the 1921 plot at Ithaca and inoculated with pure cultures. The results, given in table 6 show that the percentage of badly dwarfed plants increases with the heavier seeding of the land.

TABLE 6. EFFECT OF RATE OF SEEDING ON THE DISEASE

Rate of seeding per rod-row	Per cent of take-all-infected plants			Per cent of plants not infected
	Plants less than ½ normal height	Plants ½ to $\frac{9}{10}$ normal height	Plants of normal height	
Light, 7½ grams	23.2	30.3	46.5	0.0
Normal, 15 grams	30.0	33.5	33.5	3.0
Heavy, 30 grams	38.0	34.0	28.0	0.0

CONTROL

EXCLUSION

A quarantine to prohibit the importation of possible harborers of the pathogene was attempted in the United States in 1919 (Ausley, 1919). Present evidence, such as the general distribution of take-all in the State

of New York and its known occurrence in Oregon, Arkansas, Kansas, Indiana, Tennessee, North Carolina, and California, indicates that it has been present in this country for many years but has only recently been recognized. Since the pathogene apparently was generally present in the United States previous to the laying of the quarantine, this attempt at exclusion cannot be expected to be of any importance in the control of the disease.

ERADICATION

Investigators agree that when the take-all pathogene has once become established in an area the most practical method of control is by eradication. The methods to be employed involve the killing of the ascospores and the starving-out of the fungus.

Cultivation has for its primary object the destruction of grass and cereal harborers of the pathogene, and to accomplish this, the ground, as Dawkins (1914) states, must be kept absolutely free of any grass on which the fungus can thrive. This can best be done, according to Sutton (1911), by cultivation soon after the wheat harvest and subsequent working of the land after each rain. Other investigators, among them Gray (1914), recommend one or two whole years of fallowing. The fallowing of the ground for one or more years, as suggested for the Australian conditions, cannot be followed everywhere.

In New York the usual rotation practice makes it impossible to plow the land after the wheat harvest. Rotation must be depended upon here to starve out the fungus.

There is a considerable difference of opinion as to the required length of this rotation. If susceptible grasses, barley, and rye are not present in the rotation, the length is given by Sutton (1911) as two years, by McAlpine (1904) and Gray (1914) as three years, and by Richardson (1911) and others as from four to six years. The importance of rotation as a control measure may be inferred from the fact that during the 1921 and 1922 surveys in New York, it was observed that in nearly every field in which more than five per cent of the plants were killed by take-all, two or more successive crops of wheat had been planted. A rotation, to be effective, should have wheat not oftener than every four or five years, since the fungus can live in the soil for five months, and on the stubble as spores for from nine to ten months; and if *Agropyron repens* or other wild hosts are present, several years of cropping may be required to kill out the grass and with it the fungus.

The stubble of diseased wheat being the principal harborer of the take-all fungus, some means of disinfection, such as burning the stubble, should be applied to destroy this source of inoculum. Under Australian conditions McAlpine (1904), Richardson (1911), and Brittlebank (1920) advocate this means of disinfecting the field, but Sutton (1911) says that a stubble burn is of doubtful value since it does not destroy the under-

ground parts on which perithecia are produced. Under New York conditions the burning of stubble would kill the clover seeding which is planted with wheat, and would thus be of doubtful value. Straw, which would be likely to contain culms broken or cut off low enough to make them carriers of the organism, should be disposed of in such a way that none of the stubble or straw is returned to the land in manure or in other ways for at least three years preceding the planting of wheat. Observations in 1921 showed that wheat straw from diseased fields, when applied in manure during the fall and winter of 1920–21, acted as a direct carrier of the fungus.

PROTECTION

The first measure by which protection is to be obtained is by modifying moisture relations. Humidity of the soil has been reported as being favorable to the disease (Robinson, 1907, and Mangin, 1914), and the present investigations seem to confirm this theory. Low, wet soil should therefore be drained before the wheat is planted.

The second measure of portection is by modification of temperature relations. From the evidence presented under *Ecology*, it appears that high temperatures in the fall increase the amount of seedling infection. The planting of winter wheat as late as possible will reduce the amount of infection and the resulting amount of diseased and badly dwarfed plants at harvest time.

The third measure of protection is by modifying the chemical reaction of the soil. Brittlebank (1920) found that an alkaline soil produced more take-all than one which was non-alkaline. The present investigations show that the addition of lime increases, and the addition of acid or acid-forming substances decreases, the percentage of badly diseased plants. A study of the germination of ascospores and the rate of growth of the organism at different degrees of acidity indicated that acid added to the soil acts as a barrier between the pathogene and the susceptible roots of the host plants, in some way, probably by inhibiting the germination of the ascospores and the growth of the fungus. It was observed also that the yield of uninfected plants was reduced as the acidity increased below pH 7. The advisability of using acid on the soil to control take-all would therefore be dependent on such factors as the severity of the disease in any one locality and the degree of tolerance to acid conditions of the other crops in the rotation.

The results seem to indicate that where wheat, barley, rye, and potatoes are the only crops in the rotation, and where the disease is severe, the application of a moderate amount of an acid-forming substance would be a profitable control measure. In New York certain crops such as clover, which is intolerant to acid conditions, are often grown in the rotation. It therefore seems unlikely that the addition of acid would

be practicable here. Nevertheless, from the point of view of take-all control, the practice of adding lime to the soil before planting wheat should be discontinued.

IMMUNIZATION

From the evidence presented under *Varietal Susceptibility*, it is probable that such varieties of wheat as Forward and Dawson will be found resistant to the disease under field conditions and may be recommended for planting in areas where take-all is most severe.

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